INTRODUCTION

Morbid obesity is a chronic, life long, multifactorial congenital disorder characterized by excessive fat deposition and associated mental psychological physical social and economic problems and as a result present a significant thread to health ***(Unger et al., 2008).***

The prevalence of obesity ranges from 9% to 26%, and the prevalence of morbid obesity, which is a known risk factor for metabolic syndrome, diabetes mellitus and cardiovascular disease, ranges from 1.4% to 7% ***(Power et al., 2011).***

Man has become sedentary with computers, remote controls, taking elevators for even one floor, increased cars and high calorie convenience food ***(Buchwal et al., 2005).***

Overweight and obesity are associated with increased rate of type II diabetes mellitus, hypertension, cardiovascular diseases, dyslipidemia, arthritis, non-alcoholic steatohepatitis, gallbladder disease, sleep apnea syndrome and several cancers. Mortality increases with increasing body mass index. Mortality rate is twelve times than that in young normal weight men ***(Olshansky et al., 2005).***

Nonalcoholic fatty liver disease (NAFLD) is one of the most common forms of liver disease across the world. NAFLD represents a spectrum ranging from bland steatosis to

nonalcoholic steatohepatitis (NASH) and is closely associated with obesity and metabolic syndrome ***(Ludwig et al., 1980).***

The association between NAFLD and obesity makes weight loss a plausible treatment strategy in NAFLD patients. Substantial weight loss has been shown to have a significant benefit, but most patients fail to achieve a meaningful decrease in BMI with diet and exercise. Bariatric surgery provides the only reliable method of weight loss in severe obesity and has been increasingly available for obese patients with NAFLD ***(Blackburn et al., 2009).***

The procedure can be divided into 3 broad categories based on their mechanism of action***:***

1- Restrictive procedure, which aim to restrict the amount of food that can be eaten by surgically reducing the size of the stomach. Restrictive procedures most commonly performed are vertical banded gastroplasty, laparoscopic adjustable gastric banding and sleeve gastrectomy.

2- Malabsorptive procedures are less popular than restrictive procedures as they are more technically demanding to be performed and patients often develop nutritional deficiencies. Procedure aim to bypass a segment of the small bowel so that less food is absorbed (biliopancreatric diversion with or without duodenal pouch).

3- Hybrid procedure aim to restrict food intake by creating a small gastric pouch which also limits absorption by bypassing the proximal small bowel- the roux-en-y gastric bypass (RYGB), It leaves most of the small bowel intact and so avoids many of the unwanted malabsorptive side effects such as diarrhea and nutritional deficiencies. This is the most common surgical bariatric procedure performed. (Runkle et al., 2011).

These operations achieve a reduction of at least 50% loss of excessive weight (which include visceral fat reduction) and patient benefits from an improvement in glycemic control, lowered blood pressure and better metabolic profiles. More invasive procedures such as biliopancreatic diversion are associated with greater degrees of weight loss but have a higher complication rate. Formal trials of bariatric surgery in NAFLD are lacking. Liver histologic improvement has been documented with all techniques ***(Klein et al., 2006).***

AIM OF THE WORK

The aim of the work is to evaluate the current status, prognostic factors and results of surgical treatment of morbid obesity, with stress on its effect on morbidly obese patients with fatty liver.

OBESITY

Obesity has truly become a worldwide problem, affecting countries rich and poor. One of the most recent and careful global estimates find that roughly 500 million adult are obese (defined as a body mass index, or BMI of 30 or higher that is almost 10 per cent of men and 14 per cent of women- and it is nearly double the rate of obesity in 1980. Nearly 1.5 billion adults were overweight or obese (defined as a BMI of 25 or higher) ***(Finucane et al., 2011).***

Not that long ago, obesity was largely a problem of the rich primarily in wealthy countries. Globalization, though, has made the world wealthier. And as poor countries move up the income scale, and people shift from subsisting on traditional diets to overeating on western diets, obesity becomes a disease of the poor. The result: over the past few decades, obesity has become "pandemic" in developing countries ***(Popkin et al., 2012).***

On the paradox of the so called "nutrition transition" is that even as obesity rates rise, underweight persists, sometimes within the same household. Low- and middle- income countries often face a dual burden- the infectious disease that accompany malnutrition and, increasingly, the debilitating chronic diseases linked to obesity and western life style ***(Doak et al., 2005).***

Definition:

Obesity is characterized by excess body fat and generally defined by the body mass index (BMI), which takes into account weight and height. This index is calculated by dividing weight in kilograms by height in meters squared: it is there for expressed in kilograms per square meter (kg/m^2). The term obesity applies when the BMI is greater than or equal to $30kg/m^2$. If the BMI is between 25 and $29.9kg/m^2$, it is called overweight. According to the International obesity task force, obesity can be divided into three categories: obese class 1(BMI from30.0 to 34.9 kg/m^2), obese class 2(from35.0 to 39.9) and obese class 3(greater than or equal to 40 kg/m^2). Morbid obesity refers to obese class 3 or to obese class 2 if it is associated with other co- morbidity factors ***(WHO, 2003).***

Table 1: Weight Classification According To Body Mass Index ***(WHO, 2008).***

CLASS	BMI	Risk of co morbidities
Underweight	$<18.5kg/m^2$	Less than $18.5kg/m^2$ Low (although there may be other problems associated with being underweight)
Normal	18.5-24.9	Normal
Overweight	25-29.9	Increased
Obesity(class1)	30-34.9	Moderate
Obesity(class2)	35-39.9	Severe
Obesity(class3)	Above 40	Very sever

Obesity Co-Morbidities:

Obesity is a major cause of preventable health problems and mortality. It is strongly linked to type 2diabetes and a risk factor for many other chronic conditions such as hypertension, cardiovascular disease, some cancer and arthritis. The table below shows the health conditions associated with being obese, including problems that result from metabolic consequences of obesity and those directly related to excess weight. The level of risk increases sharply with a BMI above 35. Depression, low self-esteem, social prejudice, and reduced work opportunities can add to the burden of people who are obese ***(WHO, 2003)***.

Table 2: Disease and conditions associated with obesity ***(National Health and Medical Research Council. Canberra, 2003).***

Relative risk	Associated with metabolic consequences	Associated with excess weight
Greatly increased	Type 2 diabetes Gall bladder disease Hypertension Dyslipidemia Insulin resistance Non- alcoholic fatty liver disease	Sleep apnoea Breathlessness Social isolation and depression Daytime sleepiness and fatigue
Moderately increased	Coronary heart disease Gout/ hyperuricaemia	Osteoarthritis Respiratory disease Hernia Psychological problems
Slightly increased	Cancer(breast, endometrial, colon and others) Reproductive abnormalities Impaired fertility Polysystic ovaries Skin problems Cataracts	Varicose veins Musculoskeletal problems Back problems Stress incontinence Oedema/ cellulitis.

Pathogenesis of Obesity:

Fat accounts for 21% to 37% of weight of middle aged men and women. At some time in life, the obese individual takes more calories than he or she expended and appetite was not subsequently compensate for the increase in stored energy and this will lead to obesity ***(Campbell et al., 2005).***

Appetite stimulants include, Neuropeptides Y (NPY), Nor-epinephrine, Epinephrine, Insulin, Glucocorticoids, Galanin, Opioids (dymorphins, endorphins, and enkephalins), Growth hormone-releasing hormone, Somatostatin, polypeptide Y, Melanocortin Hormone (MCH) and Neurotensin ***(Bray et al., 2002).***

The Appetite suppressants are Serotonin, Leptin, Cholecystokinin (CCK), Corticotropin-releasing hormone (CRH), Urocortin, Glucagon, Glucagon-like peptide-1 (GLP1), Gammaaminobutyric acid (GABA) and Glucose (***La Barre et al., 2009).***

Mechanisms controlling fat cell size and numbers are still poorly understood, however, there are several factors that are known to share in the pathogenesis of obesity:

1. Lipoprotein Lipase Enzyme:

The enzyme lipoprotein lipase, produced by the adipose tissue and residing on its capillary endothelium, permits fat

cells to take up fatty acids from circulating chylomicrons (dietary fat) and very low density lipoprotein ***(Pi-Sunyer et al., 2000).***

2. Leptin Hormone:

Leptin is the best known of the afferent fat signals and the best candidate for primary signal communication of body fat information to the central controller. Identification of this peptide through positional cloning in 1994 provided major new insights into the regulation of food intake, energy expenditure, and body fat. It is now clear that this cytokine - derived primarily from fat cells, but also from the placenta and possibly the stomach - reduces food intake and increases the activity of the thermogenic components of the sympathetic nervous system ***(Farooqi et al., 2007).***

3. Ghrelin Hormone:

Ghrelin is a newly recognized gastric hormone with orexigenic and adipogenic prosperities produced primarily by the stomach. It is reduced in obesity and weight loss is associated with increased its plasma level ***(Geloneze et al., 2003).***

4. Adiponectin:

It is a protein derived exclusively from adipose tissue. It seems to have protective metabolic and ant inflammatory properties and reduces the inflammatory changes in the

cardiovascular system that leads to heart disease. It decreases with obesity and increase with weight loss ***(Campbell et al., 2005).***

5. Resistin:

It's a peptide hormone secreted by white fat cells discovered and named after resistance to insulin. Genetic studies show a relationship between obesity, insulin resistance and resistin hormone ***(Ukkola et al., 2005).***

6. Cytokines:

Obesity is associated with increased plasma levels of cytokines (inflammatory factors) such as interleukins, C-reactive protein, tumor necrosis factor and insulin like growth factor 1. These cytokines are produced by adipose tissue and are responsible for inflammatory changes in the cardiovascular and other systems ***(Campbell et al., 2005).***

7. Serotonin:

Serotonin also plays an important role in the regulation of food intake and nutrient stores. Tryptophan and 5-hydroxytryptophan, two precursors of serotonin that increase its concentration at neuro-effector junction, both decrease food intake ***(Bray et al., 2002).***

Many other proteins are in various stages of research including pro 12 Ala polymorphism, omentin, adipocyte

complement related protein, pancreatic peptide YY, uncoupling 2 and 3, neuro-peptide Y and GLP-1 and 2 all of which can be considered as energy regulators ***(Campbell and Haslam, 2005).***

Causes and Risk Factors of Obesity:

Any small disparity between energy intake and energy expenditure gradually leads to weight gain. A continued excess of energy intake over energy expenditure gradually leads to obesity ***(Wilding et al., 2006).***

The Following are Known Causes of Obesity:

1- High-Energy Intake:

Dietary changes over the past 30 to 40 years have led to proliferation of energy-dense foods rich in fat and sugar, particularly carbonated beverages. Foods high in fat do not produce satiety as well as foods rich in carbohydrate. This leads to overconsumption of food ***(Wilding et al., 2006).***

2- Physical Inactivity

Low levels of physical activity, even if caloric intake is within normal limits, may not offset intake. Use of labor-saving devices, preferences of riding in a car instead of walking, and increases in passive forms of leisure (e.g,

television, computers) have led to an obesity-prone population ***(Chang et al., 2005).***

3- Endocrine Disorders:

A- Growth hormone deficiency (GHD):

Patients with GHD have an abnormal body composition with increased body fat and decreased lean body mass. Patients are often overweight or obese with central adiposity ***(Wilding et al., 2006).***

B- Cushing's syndrome:

Weight gain is a prominent symptom in Cushing's syndrome. There is an accompanying deposition of fat in face, neck, abdomen and mediastinum ***(Wilding et al., 2006).***

C- Thyroid disorders:

Patients with hypothyroidism may show moderate weight gain because of slowed metabolism. In rare situations, the metabolic rate in hyperthyroid patients does not increase significantly; this is accompanied by disproportionate increase in appetite leading to hyperphagia and weight gain ***(Wilding et al., 2006).***

4- Drugs:

Intake of certain kinds of drugs leads to weight gain, particularly centrally acting drugs and neuroleptics. These drugs exert their effect either centrally, affecting appetite

control (e.g, neuroleptics), or peripherally (eg, hypoglycemic drugs and protease inhibitors) ***(Wilding et al., 2006).***

5- Genetics:

Single-gene defects are known to cause obesity. These may include mutations in leptin, its receptor, and the proopiomelanocortin (PMOC) gene ***(Druce et al., 2006).***

6- Hypothalamic Abnormalities:

The hypothalamus maintains energy homeostasis; tumours may cause disruption in its function. However, hypothalamic abnormalities are exceedingly rare causes of obesity ***(Peters et al., 2007).***

The following are some of the Known Risk Factors of Obesity:

1- Low Socioeconomic Status:

Obesity is linked to food insecurity, which refers to lack of food access because of low income levels ***(Martin et al., 2007).***

2- Low Education Level:

Health literacy is the ability to understand and act on health information. A person's general literacy skills reflect his health literacy abilities ***(Davis et al., 2008).***

3- Female Gender:

Prevalence of obesity in women is 34% as compared with 32% in men ***(Ogden et al., 2007).***

4- Psychological Conditions:

Studies have shown that increased weight is associated with depression, which supports a reciprocal relationship between the two conditions ***(Hrabosky et al., 2008).***

Complication of Morbid Obesity:

Obesity becomes 'morbid' when it significantly increases the risk of one or more obesity-related health conditions or serious diseases, also known as co-morbidities. Obesity-related diseases are serious, most are chronic, and they often occur in combination and will become life threatening if not treated aggressively ***(Aronne et al., 2002).***

Morbidly obese patients are classified according to area of main fat mass:

1. Peripheral (Gynecoid) Obesity: associated with degenerative joint disease and venous stasis in the lower extremities.

2. Central (android) Obesity: associated with the highest risk of mortality-related problems due to the "Metabolic Syndrome" as well as increased intra-abdominal pressure (IAP) ***(Bray et al., 2004).***

The metabolic syndrome (MS) is a cluster of metabolic risk factors including central obesity, insulin resistance, impaired glucose tolerance, type 2 diabetes mellitus, systemic hypertension and dyslipidemia ***(Encinosa et al., 2006).***

Increased IAP is probably responsible for obesity hypoventilation, venous stasis disease, pseudotumor cerebri, gastroesophageal reflux disease, stress urinary incontinence, and systemic hypertension. Central obesity is also associated with increased neck circumference and sleep apnoea ***(Flier et al., 2004).***

Central obesity is associated with a higher mortality than peripheral obesity, because of more metabolically active visceral adipose tissue than subcutaneous fat. So that there is a greater risk of type 2 diabetes, and hyperinsulinism. Increased insulin secretion is thought to increase sodium reabsorption and, thus, cause hypertension. Central obesity also is associated with a greater production of low-density lipoprotein, leading to a higher incidence of atherosclerotic cardiovascular disease ***(JAMA, 2004).***

Obesity is associated with increased risks of type 2 diabetes, hypertension, cardiovascular disease, dyslipidaemia, non-alcoholic steatohepatitis (NASH), gall-bladder disease, arthritis, sleep apnea syndrome and several cancers ***(Flier et al., 2004)***.